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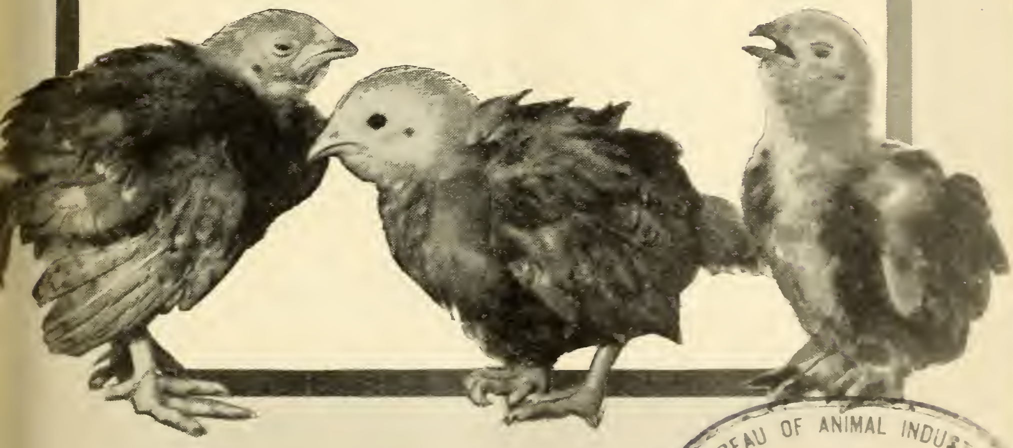
Controlling GAPEWORMS in Poultry



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CONTROLLING GAPEWORMS IN POULTRY

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Introduction

The poultry gapeworm was first discovered by Dr. Andrew Wiesenthal of Baltimore, Md., in 1797. He stated that gapeworm infection was prevalent in chickens about that city and was responsible for destroying eight-tenths of the chicks in many sections. For many years following this discovery, and before changed poultry-husbandry practices brought about a reduction in mortality, gapeworms undoubtedly destroyed large numbers of birds. Although gapeworms have not been reported as causing serious outbreaks of gapes among poultry in the United States for many years, these parasites nevertheless take a considerable toll in the form of retarded growth of chickens and some death losses. They have been reported also as causing many deaths among pheasants in some Northwestern and Eastern States.

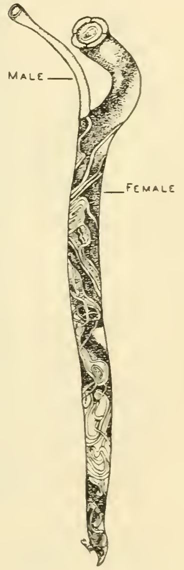

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Figure 1.—Drawing of male and female gapeworms. About four times natural size.

Cause of Gapes

Gapeworm infection, commonly known as gapes, is caused by roundworms that live in the windpipe of the bird host. As seen in the trachea, the male and female worms are jointed in permanent copula (fig. 1); for this reason they have often been referred to as forked worms.

Because of their red color, they have sometimes been called "redworms." The female is from ½ to 1 inch long and the male about ⅕ of an inch. A nodule is usually formed at the site of the attachment of the male, indicating that this worm may remain attached to the tracheal wall during its entire parasitic existence. Nodules formed in the tracheas of infested turkeys (fig. 2) and pheasants sometimes become quite large. The female worm apparently does not remain attached permanently, but loosens her hold from time to time for the purpose of selecting new feeding places.

Symptoms

Typical symptoms of gapeworm disease are usually observed in young birds. Within 1 to 2 weeks after infection, affected birds begin to gape (see cover picture) and to develop a slight hissing sound or cough which gradually becomes more severe as the disease progresses. These symptoms are usually accompanied by a quick jerk of the head forward and upward and an extension of the neck, indicating that the birds are not getting enough air. Affected birds refuse food and water, become very weak, and have difficulty in keeping up with their companions. Young birds frequently die as a result of gapeworm infection.

Young turkey poults usually develop symptoms of gapeworm infection 2 or 3 days earlier than young chickens, and die from gapeworm infections sooner.

Older birds usually show only mild symptoms or none at all. Partly grown turkeys sometimes develop pronounced symptoms of coughing which are accompanied by discharges of bloody mucus; death may occur within 2 to 3 weeks after infection. Turkeys 1 year of age may develop a deep hoarse cough which may persist for several weeks, but death rarely occurs except in cases of very heavy infections.

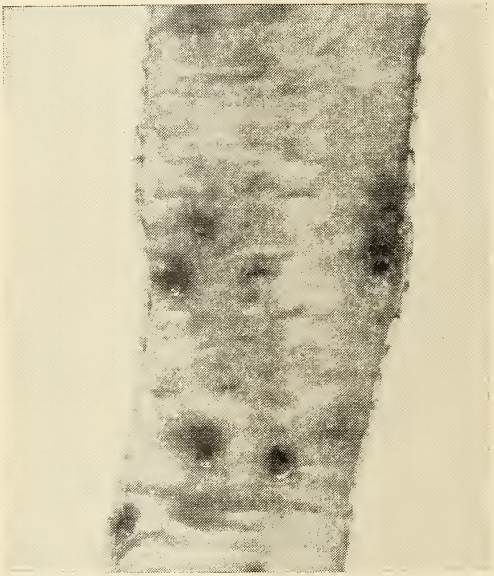
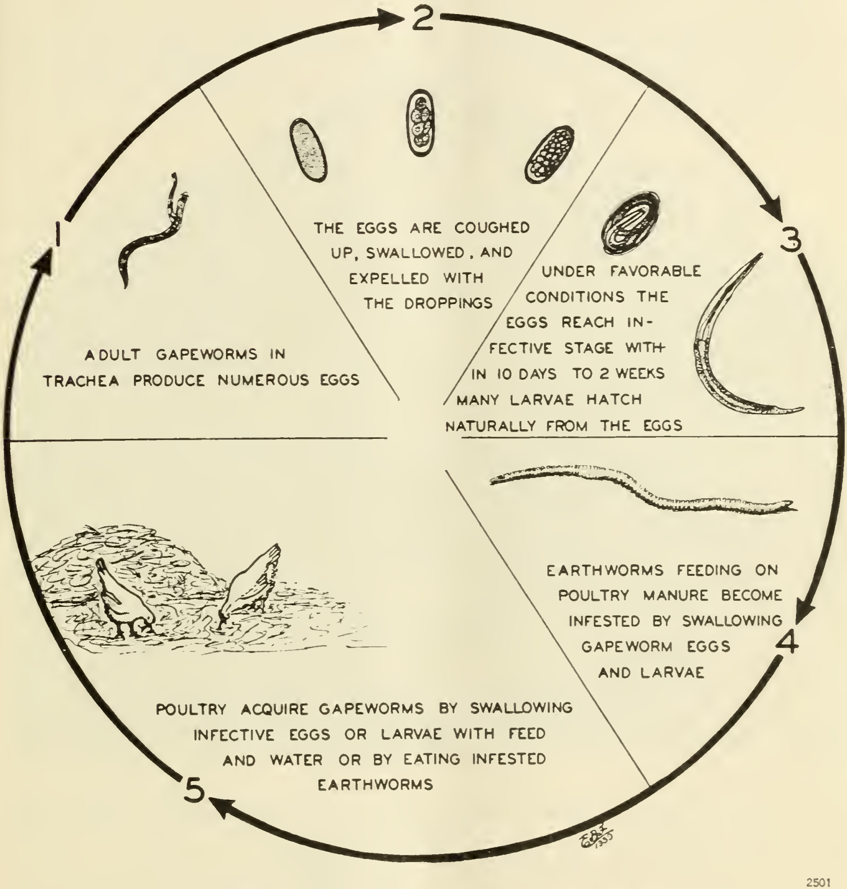


FIGURE 2.—Trachea of mature turkey showing gapeworm nodules. About natural size.

How Poultry Become Infected With Gapeworms

Poultry usually become infected with gapeworms by (1) ingesting earthworms from soil contaminated with the droppings of infected birds, or (2) swallowing with the feed and water gapeworm eggs, containing infective larvae, or (3) swallowing the larvae which have hatched from the gapeworm eggs. It was a common belief many years ago that fowls became infected by ingesting eggs contained in fresh droppings or in freshly coughed-up worms. Recent investigations have shown definitely that such eggs, when swallowed by a susceptible bird host, do not produce an infection and that it is necessary for the eggs to remain outside the body of the bird host for at least 9 to 14 days before becoming infective. As a rule, a number of the infective eggs hatch, and the larvae live free in the soil. Infection may take place when such larvae are swallowed by a susceptible host.

Figure 3 illustrates the life cycle of the gapeworm and shows that the eggs produced by the gapeworms in the tracheas of birds develop outside the host. The figure shows, as previously stated, that the birds become infected by swallowing the developed gapeworm eggs or free larvae, or by swallowing earthworms that have ingested such eggs or larvae with soil contaminated with the droppings of infected birds.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ADULT GAPEWORMS IN TRACHEA PRODUCE NUMEROUS EGGS
THE EGGS ARE COUGHED UP, SWALLOWED, AND EXPELLED WITH THE DROPPINGS
UNDER FAVORABLE CONDITIONS THE EGGS REACH INFECTIVE STAGE WITHIN 10 DAYS TO 2 WEEKS
MANY LARVAE HATCH NATURALLY FROM THE EGGS
EARTHWORMS FEEDING ON POULTRY MANURE BECOME INFESTED BY SWALLOWING GAPEWORM EGGS AND LARVAE
POULTRY ACQUIRE GAPEWORMS BY SWALLOWING INFECTIVE EGGS OR LARVAE WITH FEED AND WATER OR BY EATING INFESTED EARTHWORMS
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FIGURE 3.—Life history of poultry gapeworm.

Young Birds Most Severely Affected

Turkey poults, goslings, young chickens and young pheasants are very severely affected by gapeworms. As the windpipe of a young bird is relatively small and only a few worms are necessary to clog the opening, death by suffocation is usually the result. Guinea fowls are susceptible to gapeworm infection at any time during their lives but manifest no visible symptoms.

Mature chickens rarely become infected with gapeworms. Should they become infected, the worms seldom remain in the windpipe longer than a month. Mature chickens, therefore, have not been considered an important factor in the spread of gapeworm disease. Ducks and pigeons have been reported as hosts of the poultry gapeworm, but all attempts by the writer to infect adult birds experimentally have been unsuccessful.

Results of investigations have shown that both turkeys and guinea fowls are susceptible to gapeworm infection at any age, the former remaining infected for 3 to 7½ months and the latter for only 2 to 3 months. Adult turkeys, therefore, play an important role in the dissemination of gapeworm disease.

Wild Birds as Hosts of the Poultry Gapeworm

In the United States natural infections with the poultry gapeworm have been reported as occurring in the ruffed grouse, quail, and pheasant. Artificial infections have been produced in English sparrows and starlings. Gapeworm infection apparently causes little or no distress to ruffed grouse and quail under natural conditions, but numerous reports of high mortality among pheasants have been received from many sections of this country, particularly from the Northwestern and Eastern States.

In Europe the poultry gapeworm occurs naturally in the starling, rook, jackdaw, red grouse, and other wild birds. European investigators were not able to infect domestic birds with the wild bird strain with any degree of success until the earthworm was used as an intermediate host. For this reason, doubt has been expressed regarding the role that wild birds play in the spread of gapeworm disease in Europe.

Prevention

Raising young birds separate from the older birds and on areas not previously occupied by gapeworm-infected birds is one of the most important preventive measures. In case sufficient land is available for poultry runs, land on which infected birds have not ranged for at least 2 years may be used without immediate danger of losses from gapeworm disease. Sandy, well-drained soil is preferable, since such soil is unfavorable for the development of gapeworm eggs and larvae and is less likely to be inhabited by earthworms.

The division of a large poultry enterprise into several flocks or units is a sound practice, since outbreaks of the disease may be confined to relatively smaller areas, thus expediting a quicker clean-up.

The removal of droppings at intervals of at least a week and the disposal of such droppings in a manner that birds have no access to them aids materially in controlling gapeworm disease by eliminating the chief source of infection. Deep burial or burning of the droppings is preferred from a disease-control standpoint but, for practical reasons, other methods that utilize the fertilizing elements in the manure may be more desirable.

Treatment

Mechanical devices, such as a small quill feather stripped of all its web, except a small tuft at the end, a horsehair bent in the middle with the two ends twisted together so as to form a loop, and wire extractors have been advocated in the past for the removal of gapeworms. These devices have proved to be slow, expensive, unreliable, and rather dangerous.

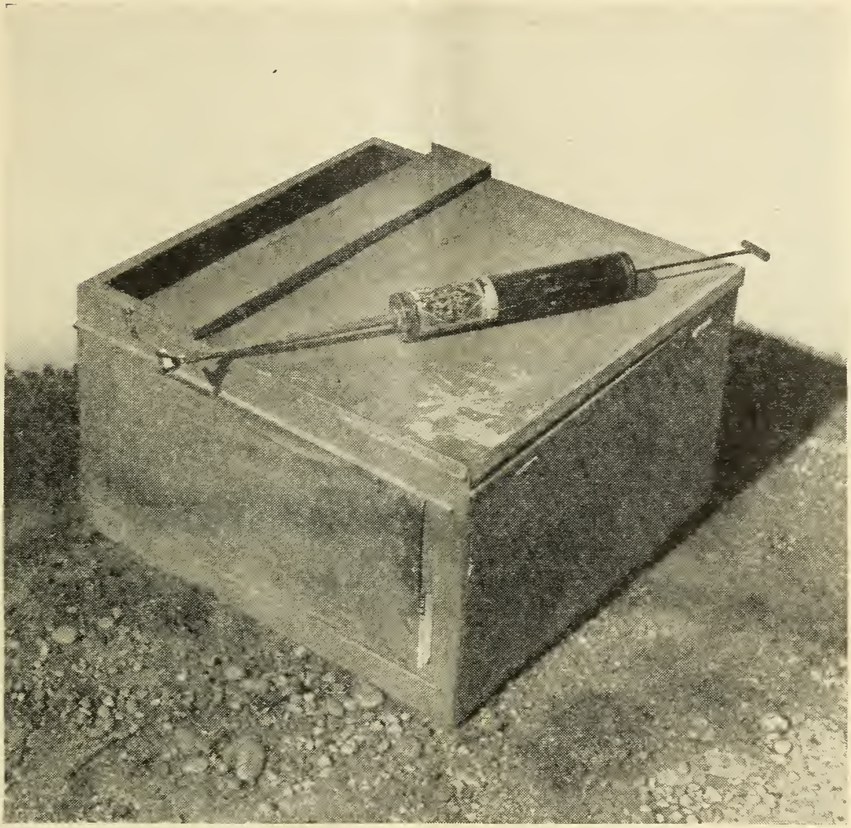
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FIGURE 4.—Dust gun and box used for treating young chickens for gapeworms.

Various drugs have been recommended, but none has proved to be as satisfactory, in controlled tests, as barium antimonyl tartrate. Investigations conducted recently by the Bureau of Animal Industry have shown that this new compound, barium antimonyl tartrate, when inhaled by the birds in the form of a dust, is effective in removing gapeworms. For treatment, the infected birds are placed in a closed container or box (fig. 4) and exposed to the powder for 15 to 20 minutes. The size of the dose is determined by the cubic capacity of the container. One ounce of barium antimonyl tartrate is sufficient for a box having a capacity of 8 cubic feet. The box should be deep enough to allow a space of at least 6 inches above the heads of the birds when standing erect. In the beginning, one-third of the total dose is blown into the box by means of a dust gun through an opening at the top. The box, if of a convenient size, is then tilted slowly from one side to the other several times. Tilting causes the birds to stir around in the box, thereby aiding in redispersing any powder that may have settled on the feathers or the floor of the box, and forcing the birds to breathe more heavily and more frequently. This affords a better opportunity for the powder to reach the worms that may be located in the lower part of the windpipe. In the case of mature birds, when the treatment box is likely to be too heavy to tilt, a small electric fan may be placed on the floor of the box to keep the powder agitated. Five minutes after the introduction of the first one-third of the powder, the second one-third is introduced, and the tilting or the use of the fan is repeated. The remaining powder is introduced 10 to 15 minutes after the beginning of the treatment, and the box is again tilted or the fan used. The birds are released 5 to 10 minutes after the last of the powder has been blown into the box.

Barium antimonyl tartrate is available commercially. Directions for making this powder, which should be prepared by a druggist or chemist, are as follows:

To prepare approximately 1¾ pounds of barium antimonyl tartrate, dissolve 244.3 grams of crystallized barium chloride in 800 cubic centimeters of warm distilled water having a temperature of 70° to 75° C. (158° to 167° F.), and mix this solution slowly with a warm solution of potassium antimonyl tartrate made by dissolving 667.8 grams of the crystalline substance in 1,700 cubic centimeters of distilled water The mixture is then stirred vigorously with a glass rod. A precipitate forms and is collected by filtering immediately through paper with the aid of a suction filter; the precipitate collected in this manner is washed with cold water and then with alcohol to facilitate drying. The drying is carried out at room temperature, since higher temperatures may cause decomposition while the chemical is wet. When properly prepared, the product will pass through a 100-mesh sieve. Otherwise it must be ground to that degree of fineness.

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